quadrigemina; the brain arteries were normal, and he designates the case as one of encephalitis.

Jürgens has described a case in which at death there were signs of secondary encephalitis. At the autopsy there was found an acute universal encephalitis. He regarded the disease as exceedingly rare.

The most complete description we have found is a case of Bechterew's.[12] This case in its histology resembles our own case very closely. The course was somewhat longer. The first symptoms developed in the year following infection, and death occurred two years later. He names the case one of disseminated syphilitic focal sclerosis, and adds that it is the only case of this type which has been completely studied, both clinically and pathologically.

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SOME OBSERVATIONS ON THE EFFECT OF ALTERNATING CURRENTS OF MODERATE FREQUENCY ON DOGS.

By G. W. Crile, PhD., M.D.,
PROFESSOR OF SURGERY,
AND
J. J. R. Macleod, M.B., D.P.H.,
PROFESSOR OF PHYSIOLOGY, WESTERN RESERVE UNIVERSITY, CLEVELAND, OHIO.

The extensive and accurate researches of Prevost and Battelli[1] leave little to be investigated as to the effects which follow, and the cause of death from the application of commercial electrical currents to animals. We think it desirable, however, that we place on record some observations which we have had the opportunity of making on the effect of alternating currents of high frequency on anæsthetized dogs; the more so, since we believe that the observations throw some light on the cause of the variable lethal effect of such currents.[1]

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[1] Journal de physiologie et de pathologie générale (1) 1899, tome i. p. 399; (2) p. 427; (3) p. 689 (4) 1900, tome ii. p. 755; (5) 1901, tome iii. p. 1085; (6) p. 1144.

Previous to Prevost and Battelli's papers considerable confusion existed as to the exact cause of death from electrical currents. d'Arsonval[2] and Kratter[3] ascribed death to respiratory cessation from paralysis of the bulbal centre; whereas, Tatum[4] and Biraud,[5] thought it to be due to arrest of the heart. Oliver and Bolam[6] came to the conclusion that currents of 200 volts act directly on the heart (*i. e.*, not through the vagal mechanism), but do not affect the respirations; with stronger currents, on the other hand, the heart and respiratory centre are simultaneously affected. Most of these workers further observed that the effect of the current and its lethality varies considerably for different groups of animals. Cunningham[7] concluded that fibrillary contraction of the heart was the usual cause of death.

Prevost and Battelli found that alternating currents of low tension (about 130 volts) cause only temporary arrest of respiration, but that fibrillary contraction of the ventricle is induced exactly similar to that which follows the application of a faradic current directly to the heart. In dogs these fibrillations always persist after the current is broken, so that, once the fibrillations have appeared, it is impossible to resuscitate the animal. In guinea-pigs and rabbits the fibrillations may sometimes disappear and the animal recover; whereas, in rats this latter result was invariably noted, this animal being apparently absolutely immune to low-tension currents. With very high-tension currents (1300 to 4800 volts), Prevost and Battelli[8] found that fibrillation of the ventricle does not occur, but rather that the beats become quicker and stronger, and that the arterial blood pressure rises. The respiratory centre, however, becomes paralyzed, and profound shock results. This respiratory paralysis could frequently be recovered from by the application of artificial respiration; and in dogs spontaneous reappearance of respiration was quite common. It was further noted that when the heart had come to a standstill—coincidently with respiratory cessation—massage of it brought on fibrillation. Still another most interesting observation deserves notice, namely, that the application of a high-tension current to a heart already in fibrillary contraction causes the fibrillations to disappear and the normal heart-beat to be re-established.

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[1] These observations were concluded a year ago, but the publication was withheld in the hope of our being able to prosecute the investigation farther. This we find now to be impossible.

[2] Prevost and Battelli. Loc. cit., No. 1.

[3] Der Tod durch Electricität, Leipzig, 1896.

[4] Death from Electrical Currents, New York Medical Journal, 1890, vol. lv.

[5] Prevost and Battelli. Loc. cit., No. 1.

[6] On the Cause of Death by Electric Shock, British Medical Journal, 1898, p. 132.

[7] A Text-book of Legal Medicine and Toxicology, Philadelphia, 1903, p. 261.

[8] Loc. cit., No. 2.

The other papers of Provost and Battelli refer to continuous currents (No. 3) and to the influence of the number of periods of the alternating current on its physiological effect.[1] In this latter research it was found that with very frequent alternating currents—*e. g.*, 1720 periods per second, a much higher voltage is necessary to produce death than when currents of low frequency (*e. g.*, from 9 to 150 periods) are used. It is also pointed out in this paper that the respirations, suspended during the application of the current, spontaneously reappear when the current is broken, provided the heart be not paralyzed. The other papers published in 1899[2] refer to the physiological effect of electrical discharges from condensers. Since these papers, others have appeared by Arloing,[3] Bordier et Lecomte,[4] and Guilnard,[5] but as there is nothing in any of these regarding the effect of alternating currents on dogs, a further reference to them will be unnecessary here.

Practically all the experiments carried out by us were with alternating currents, as used for illuminating purposes. In strength these varied between 400 and 2700 volts; in frequency sixty periods per second. In all cases dogs, rendered anæsthetic by morphine and A. C. E. mixture, were employed. The electrodes were made of copper or zinc, shaped either as thick rods, which could be placed in the rectum and mouth, or spoon-shaped, to apply to the surface of the body. The electrodes were usually wrapped around with a sponge soaked in saline solution, so as to increase the conductivity. The carotid blood pressure was recorded in most cases, and the respirations carefully observed.

The results of the experiments are summarized in the appended tables:

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[1] Prevost and Battelli. Loc. cit., No. 4.

[2] Loc. cit., Nos. 5 and 6.

[3] Contribution a la connaisance de l'action des currents électriques continus a haut voltage sur les chevaux, Journ. de physiol. et de path. génér., 1902, tome iv. p. 976.

[4] Effets de l'application directe des courants de haut frequence sur les animaux, ibid., 1903, tome v. p. 564.

[5] Contributionàl étude des effets mortels produits par les courants électriques, ibid., p. 363.

No.	Strength of current (volts).	Position of electrodes.	Duration of application.	Behavior (general) of heart and blood-pressure.	Behavior of respirations.	Lethal effect.	Remarks.
1	2300	Mouth and rectum.	5 sec.	Fall to abscissa.	Cessation.	Death.	Artificial respiration and massage of heart without effect; adrenalin intravenously.
2	1000	"	17 "	"	"	"	Artificial respiration and massage of heart without effect; adrenalin intravenously.
3	2300	"	Instantaneous.	Momentary inhibition.	Unaffected.	Recovered.	Same dog used as in 3*a*.
3*a*	2300	"	"	Fall to abscissa.	Cessation.	Death.	Artificial respiration and massage of no avail; adrenalin intravenously.
4	2300	"	15 sec.	"	"	"	1 mg. atropin sulphate injected previous to application of current.
5	2300	Back of head and hind limb.	Instantaneous.	Momentary inhibition.	Unaffected.	Recovered.	Same dog used in 5*a* to 5*d*.
5*a*	2300	"	"	Marked rise.	"	"	1 mg. atropin sulphate injected previous to application of current
5*b*	2300	"	1 sec.	"	"	"	
5*c*	2300	"	5 "	"	"	"	
5*d*	2300	"	20 "	Fall to abscissa.	Cessation.	Death.	
6	2300	"	Instantaneous.	Momentary inhibition.	Unaffected.	Recovered.	Same dog used in 6*a* to 6*c*.
6*a*	2300	"	4 sec.	Momentary inhibition, then rise.	"	"	
6*b*	2300	"	5 "	Momentary inhibition, then irregular heart action; became regular on massaging.	Cessation, but recovered by artificial respiration.	"	Artificial respiration and heart massage resulted in recovery of normal heart-beat and respirations; this is the only result of this nature that was obtained.
6*c*	4000	"	5 "	Fall to abscissa.	Cessation.	Death.	The resistance of the dog had been depress'd by previous applications.
7	2300	"	2 "	Inhibition, then rapid beat.	Cessation, then recovery.	Recovered.	
7*a*	2300	"	10 "	Temporary inhibition.	Cessation, then recovery.	"	1 mg. atropin sulphate given previous to application of current.
7*b*	2300	"	10 "	Fall to abscissa.	Cessation.	Death.	Tissues at point of application of electrodes burnt; heat rigor of abdominal muscles.
8	2300	"	3½"	Fall to abscissa.	"	"	1 mg. atropin intravenously before electrocution; artificial respiration, heart massage and adrenalin injection without effect; P. M., auricles beating, venricles in diastole.
9	2300	Leg and mouth.	2 "	Fall to abscissa.	"	"	Same measures as in 8 adopted without effect.
10	2300	Not recorded.	3 "	No effect.	Unaffected.	Recovered.	5 mg. atropin intravenously before electrocution.
11	2300	Head and leg.	Instantaneous.	Momentary fall.	"	"	Both vagi cut.
11*a*	2300	"	4 sec.	Fall to abscissa.	Cessation.	Death.	3 mg. atropin injection four minutes before application of current. 2.5 amperes passed through body; artificial respiration, adrenalin injection, etc., without effect.

No.	Strength of current (volts).	Position of electrodes.	Duration of application.	Behavior (general) of heart and blood-pressure.	Behavior of respirations.	Lethal effect.	Remarks.
13	1500 1 ampere	Head and leg.	2 sec.	Fall to abscissa.	....	Partial recovery.	Artificial respiration and heart massage caused partial recovery of heart-beat and respirations, and dog came partially out of anæsthetic; readministration of anæsthetic (ether) caused death.
14	400	Head and foot.	Instantaneous.	Momentary inhibition.	Temporary cessation.	Recovered.	5 mg. atropin injected after application of current.
14*a*	600	"	"	Fall to abscissa.	Cessation.	Death.	Marked fibrillary contraction of heart.
15	800	"	"	Momentary inhibition.	....	Recovered.	6 mg. atropin injected previous to application of current.
15*a*	800	"	"	Momentary inhibition.	....	"	
15*b*	2300	"	"	Fall to abscissa.	Cessation.	Death.	Heart in marked fibrillary contraction; direct massage increased this
16	1000	"	"	....	....	Recovered.	No tracing taken; dog allowed to recover from anæsthetic; was in every way normal next day.
17	600	"	"	....	....	Death.	Marked fibrillary contraction.
18	400	On isolated heart.	....	Fibrillation	....	......	Heart perfused by Langendorff's method (see text, p. 422).
19	400	On heart in situ.	6 sec.	Fibrillation	....	......	Perfusion of heart after fibrillations had appeared of no effect (see text, p. 422).
20	2700	Hind leg and head.	4½ "	Inhibition.	Cessation.	Death.	Window in thorax; heart quickly excised and perfused by Langendorff's method; auricles began to beat and ventricles to show fibrillations with occasional beats.
21	1500	....	Instantaneous.	No effect.	No effect.	......	
22	1000	Head and leg.	"	Momentary inhibition.	....	Recovered.	Window in thorax.
22*a*	1000	"	"	Inhibition, then fibrillation.	....	Death.	
23	1000	"	"	Inhibition, then fibrillation.	....	"	Massage of heart; perfusion through coronary arteries with oxygenated Locke's fluid, anelectrotonus had no effect on fibrillary contractions.

The most noteworthy points of interest are: Firstly, the variable strength of current necessary to cause death; and secondly, the effect of the current on the circulatory system. By the study of the latter a satisfactory explanation is, we think, obtained of the cause of death in all cases.

With regard to the strength of current necessary to cause death, it will be noticed that this depends largely on the position of the electrodes. With these in the mouth and rectum death occurred after a momentary application of 1000 volts (No. 2[1]); whereas, when they were placed on the head and hind leg a current of 2300 volts, repeatedly applied for periods of from one to ten seconds, did not cause death (Nos. 5, 6, 7, and 10). Even when the currents were similarly applied (viz., to back of head and hind leg), however, currents of equal voltage had a variable lethal effect; thus, in several cases 2300 volts momentarily applied caused death (Nos. 9 and 15), and this was also induced in some by a much smaller voltage (Nos. 14 and 17). After we have considered the effect produced on the blood pressure, we will be in a position to offer a satisfactory explanation of this irregularity in lethal effect.

The effect on the blood pressure is very striking; at first there is often a slight rise, then a distinct fall to below the abscissa, and then, if the current be only of short duration, a rise again to above normal. If the current be of longer application, however, the blood pressure does not recover. The temporary rise at the moment of application is probably due to muscular contraction and perhaps vasoconstriction. The inhibition which follows is due, in some cases at least, to stimulation of the vagus mechanism, for it could be prevented in one case by the administration of atropin (No. 5). Even when atrophinized, however, or when both vagi were cut (No. 11), this inhibition, with subsequent recovery, was not infrequently noticed, and in all cases where a strong current was applied for long enough (five to ten seconds), permanent fall of the blood pressure was observed, even when the animal was deeply atropinized. Obviously, then, vagal inhibition of the heart has nothing to do with the permanent fall of the blood pressure. By a direct examination of the heart, the cause of this permanent fall of blood pressure was found to be fibrillary contraction of the ventricle, the auricles, meanwhile, continuing to beat normally (Nos. 8, 11, 14, 15, 18, 19, 20). By directly applying the electrodes to the heart, the mode of onset of this condition was more closely studied. It was found that when the current was closed, there was entire inhibition of the ventricle, and that when the current was broken the fibrillary twitchings started at the base. A current of one volt directly applied to the heart was sufficient to induce the fibrillation.

In one experiment we excised the heart and perfused it through the coronary arteries with defibrinated blood by Langendorff's method. When the beats had become normal a momentary current of 400 volts was applied to the base. The normal beat was immediately replaced by fibrillary twitchings, which persisted until the heart was quite dead.

In another experiment, after producing fibrillary twitchings with the heart *in situ*, it was quickly excised and perfused by Langendorff's method; in about two minutes normal contractions of the ventricles reappeared at the rate of 90 beats per second, but the fibrillary twitchings still persited.

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[1] These numbers refer to the table.

Direct massage of the heart *in situ* was also frequently attempted, but without avail. Anelectrotonus, produced by using various strengths of voltaic current, was likewise without effect on the condition.

We had not an opportunity of applying a very powerful and rapid alternating current, which is stated by Prevost and Battelli (loc. cit., No. 2) to arrest the fibrillation, nor did we cool the ventricle as practised by Porter[1] to dissipate the fibrillations produced by ligature of the coronary arteries. With the strongest current at our disposal (viz., 2700 volts, No. 20), applied for 4.5 seconds through electrodes placed on the head and hind limb, we noticed that the heart was merely inhibited, fibrillation not appearing until after the heart had been excised and massaged by hand, in point of time, eleven minutes after the current had been applied.

The respiratory movements were invariably brought to a standstill during the application of the current, on account of the tonic convulsions coincidently induced. They reappeared, however, when the current was broken, provided this was only of so short duration that no fibrillary contraction of the heart existed. Whenever fibrillary twitching had occurred, however, the respirations remained permanently inhibited, and this was true, even after vigorous artificial respiration.

The application of a current of 1000 volts for an instant through electrodes placed on the head and hind limb does not seem to permanently damage the nervous tissues, for in one dog (No. 16) in which this was done and the animal allowed to recover from the anæsthesia, no change from his normal behavior was perceptible (the reflexes were normal; there was no paralysis, etc.).

Coming now to the question of variability in the strength of current necessary to cause death, we are convinced that this depends on the path through the body traversed by the current.[2] If the heart lies in this path, fibrillary contractions will result; if not so, only vagal inhibition. Currents passing between electrodes in the mouth and rectum necessarily traverse the heart and are always fatal. With the electrodes on the head and hind limb, on the other hand, most of the current will pass along the tissue about the spinal column and will frequently pass by the heart.

In several experiments we placed the electrodes on the head and anterior extremities, and invariably noted vagal inhibition of the heart, general convulsions, and respiratory cessation, but no delirium cordis. The respirations returned, and the heart started beating when the current was broken.

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[1] American Journal of Physiology, 1898; vol. 1. p. 71.

[2] This explanation was first brought forward by Tatum (loc. cit.), and Cunningham, A Textbook of Legal Medicine and Toxicology, Philadelphia, 1903, p. 247.

In other experiments we placed the electrodes on the two anterior extremities, with results similar to those obtained in the previous cases, except in one dog in which fibrillary contractions followed the application of a current of 2300 volts for about ten seconds. Some of the current in this case had undoubtedly spread to the heart.

These observations seem to us to suggest *a possible prophylactic measure* for workers exposed to the danger of strong currents; viz., that they should wear a corset made of some conducting material; for example, copper. This corset, to be of any value, would require to be closely applied to the skin about the shoulders and base of the neck above, and to the lower portion of the trunk below; and opposite the cardiac region it would require to be separated from the skin by some non-conducting material—for example, India-rubber. It would certainly be of no use merely to wear such a corset separated from the skin along all its length by woollens, as, we are told, has been practised.

We are greatly indebted to the Cleveland Electric Illuminating Company for supplying us with the necessary current, and to Mr. E. E. Noble, their superintendent, who manipulated the apparatus for us.

THE PHYSIOLOGICAL RELATIONSHIP OF THE PROTEIDS OF THE BLOOD PLASMA.

By Thomas Stotesbury Githens, M.D.,
of Philadelphia.

Although it is evident that there must be wide variations in the composition of the blood plasma even in health and that this variation must affect the proteid element of that fluid, still, owing to the great technical difficulties in the separation and estimation of these bodies, it is only within recent years that their study has been taken up vigorously, and so far the work on them has been almost entirely experimental, our knowledge of the subject not being sufficient to make it of clinical use. I was able to make the series of experiments on the variations of the proteids of the plasma under varying conditions, which form the basis of this paper, in the laboratory of Professor Franz Hofmeister, in Strasburg, whom I wish to thank for his constant help during the course of the investigations.

The separation of the proteids of the plasma was very inaccurate and unsatisfactory until the method of salting out was developed. This method is based on the fact that the addition of certain salts to solutions of proteids renders these insoluble. The various proteids of blood plasma are precipitated at different concentrations, and by adding varying amounts of certain salts at least four dis-